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NEWS 7 MAY 19 Derwent World Patents Index to be reloaded and enhanced
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NEWS 10 JUN 02 The first reclassification of IPC codes now complete in
INPADOC
NEWS 11 JUN 26 TULSA/TULSA2 reloaded and enhanced with new search and
and display fields
NEWS 12 JUN 28 Price changes in full-text patent databases EPFULL and PCTFULL
NEWS 13 JUL 11 CHEMSAFE reloaded and enhanced
NEWS 14 JUL 14 FSTA enhanced with Japanese patents
NEWS 15 JUL 19 Coverage of Research Disclosure reinstated in DWPI
NEWS 16 AUG 09 INSPEC enhanced with 1898-1968 archive
NEWS 17 AUG 28 ADISCTI Reloaded and Enhanced
NEWS 18 AUG 30 CA(SM)/Caplus(SM) Austrian patent law changes

NEWS EXPRESS JUNE 30 CURRENT WINDOWS VERSION IS V8.01b, CURRENT
MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP),
AND CURRENT DISCOVER FILE IS DATED 26 JUNE 2006.

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* * * * * STN Columbus * * * * *

FILE 'HOME' ENTERED AT 13:39:33 ON 31 AUG 2006

=> file caplus

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0.21

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FILE LAST UPDATED: 30 Aug 2006 (20060830/ED)

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=> s somatostatin or neurotensin or penetratine or bombensin

19356 SOMATOSTATIN
146 SOMATOSTATINS
19365 SOMATOSTATIN
(SOMATOSTATIN OR SOMATOSTATINS)
4752 NEUROTENSIN
27 NEUROTENSINS
4755 NEUROTENSIN
(NEUROTENSIN OR NEUROTENSINS)
0 PENETRATINE
1 PENETRATINES
1 PENETRATINE
(PENETRATINE OR PENETRATINES)
1 BOMBENSIN

L1 23282 SOMATOSTATIN OR NEUROTENSIN OR PENETRATINE OR BOMBENSIN

=> s acridine or porphyrin or ellipticine or phenantroline or carbazole or benzimidazole or daunorubicine or epirubicine or mixoxantrone

17981 ACRIDINE
1711 ACRIDINES
18378 ACRIDINE
(ACRIDINE OR ACRIDINES)
35685 PORPHYRIN
24812 PORPHYRINS
41961 PORPHYRIN
(PORPHYRIN OR PORPHYRINS)
1033 ELLIPTICINE
147 ELLIPTICINES
1057 ELLIPTICINE
(ELLIPTICINE OR ELLIPTICINES)
171 PHENANTROLINE
5 PHENANTROLINES
174 PHENANTROLINE
(PHENANTROLINE OR PHENANTROLINES)
16646 CARBAZOLE
2183 CARBAZOLES
17214 CARBAZOLE
(CARBAZOLE OR CARBAZOLES)
23371 BENZIMIDAZOLE
5898 BENZIMIDAZOLES
24718 BENZIMIDAZOLE

(BENZIMIDAZOLE OR BENZIMIDAZOLES)
42 DAUNORUBICINE
16 EPIRUBICINE
0 MIXOXANTRONE
L2 102010 ACRIDINE OR PORPHYRIN OR ELLIPTICINE OR PHENANTROLINE OR CARBAZO
LE OR BENZIMIDAZOLE OR DAUNORUBICINE OR EPIRUBICINE OR MIXOXANTR
ONE

=> s 12 and 12

L3 102010 L2 AND L2

=> s 12 and 11

L4 53 L2 AND L1

=> s conjugat? or coupl? or link? or combin?

225632 CONJUGAT?

783227 COUPL?

466608 LINK?

1115681 COMBIN?

L5 2438342 CONJUGAT? OR COUPL? OR LINK? OR COMBIN?

=> s 15 and 14

L6 29 L5 AND L4

=> s 16 not py>1999

7078308 PY>1999

L7 1 L6 NOT PY>1999

=> d ibib

L7 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 1989:625888 CAPLUS

DOCUMENT NUMBER: 111:225888

TITLE: Enprostil reduces the increase of gastric corpus
mucosal mass induced by the hydrogen-potassium-
stimulated adenosine triphosphatase inhibitor BY
831-78 in the rat

AUTHOR(S): Inauen, W.; Rohner, C.; Koelz, H. R.; Herdmann, J.;
Schuerer-Maly, C. C.; Varga, L.; Halter, F.

CORPORATE SOURCE: Gastrointest. Unit, Univ. Hosp., Bern, 3010, Switz.

SOURCE: Gastroenterology (1989), 97(4), 846-52

CODEN: GASTAB; ISSN: 0016-5085

DOCUMENT TYPE: Journal

LANGUAGE: English

=> d abs kwic

L7 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2006 ACS on STN

AB It was determined if enprostil, a synthetic PGE2 derivative, might inhibit
gastrin

release and the trophic effects on gastric oxyntic mucosa induced by
prolonged treatment with an inhibitor of H⁺-K⁺-stimulated ATPase, the
substituted benzimidazole BY 831-78. Rats were treated
intragastrically with enprostil (1 or 15 µg/kg b.i.d.), BY 831-78 (15
µmol/kg once daily), the combination of enprostil and BY
831-78, ranitidine (300 µmol/kg b.i.d.), and placebo. Plasma gastrin
and somatostatin levels and gastric acid secretion were measured
during a 1-day treatment in animals fitted with chronic gastric fistulas
and repeatedly during 9 wk of treatment in intact rats. Despite
inhibiting acid secretion, enprostil did not increase plasma gastrin.
When combined with BY 831-78, enprostil transiently reduced the
BY 831-78-induced increase of integrated plasma gastrin (1375 vs. 2137
pmol/L.12h) in fasted rats with fistulas, but failed to prevent the marked
hypergastrinemia following 9 wk of treatment with BY 831-78 (717 vs. 731

pmol/L) in intact rats. However, enprostil reduced the BY 831-78-induced increase of oxyntic mucosal volume (458 vs. 567 mm³), whereas BY 831-78 prevented the enprostil-induced increase of antral mucosal volume (42 vs. 56 mm³). Apparently, some of the trophic effects induced by a H⁺,K⁺-ATPase inhibitor are not exclusively governed by gastrin.

AB . . . and the trophic effects on gastric oxyntic mucosa induced by prolonged treatment with an inhibitor of H⁺-K⁺-stimulated ATPase, the substituted benzimidazole BY 831-78. Rats were treated intragastrically with enprostil (1 or 15 µg/kg b.i.d.), BY 831-78 (15 µmol/kg once daily), the combination of enprostil and BY 831-78, ranitidine (300 µmol/kg b.i.d.), and placebo. Plasma gastrin and somatostatin levels and gastric acid secretion were measured during a 1-day treatment in animals fitted with chronic gastric fistulas and repeatedly during 9 wk of treatment in intact rats. Despite inhibiting acid secretion, enprostil did not increase plasma gastrin. When combined with BY 831-78, enprostil transiently reduced the BY 831-78-induced increase of integrated plasma gastrin (1375 vs. 2137 pmol/L.12h) in fasted. . .

IT 51110-01-1, Somatostatin
RL: BIOL (Biological study)
(secretion of, ATPase inhibitor and PGE2 analog effect on, gastrin in relation to)

=> s 16 not py>2000
6188416 PY>2000
L8 2 L6 NOT PY>2000

=> s 18 not 17
L9 1 L8 NOT L7

=> d ibib abs kwic

L9 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2000:690483 CAPLUS
DOCUMENT NUMBER: 133:361093
TITLE: Ligand-induced internalization of neurotensin
in transfected COS-7 cells: differential intracellular
trafficking of ligand and receptor
AUTHOR(S): Vandebulcke, Franck; Nouel, Dominique; Vincent,
Jean-Pierre; Mazella, Jean; Beaudet, Alain
CORPORATE SOURCE: Montreal Neurological Institute, McGill University,
Montreal, QC, H2A 2B4, Can.
SOURCE: Journal of Cell Science (2000), 113(17), 2963-2975
CODEN: JNCSAI; ISSN: 0021-9533
PUBLISHER: Company of Biologists Ltd.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The neuropeptide neurotensin (NT) is known to be internalized in a receptor-mediated fashion into its target cells. To gain insight into the mechanisms underlying this process, we monitored in parallel the migration of the NT1 neurotensin receptor subtype and a fluorescent analog of NT (fluo-NT) in COS-7 cells transfected with a tagged NT1 construct. Fluo-NT internalization was prevented by hypertonic sucrose, potassium depletion and cytosol acidification, demonstrating that it proceeded via clathrin-coated pits. Within 0-30 min, fluo-NT accumulated together with its receptor in Acridine Orange-pos., acidic organelles. These organelles concentrated transferrin and immunostained pos. for rab 5A, therefore they were early endosomes. After 30-45 min, the ligand and its receptor no longer colocalized. Fluo-NT was first found in rab 7-pos. late endosomes and later in a nonacidic juxtanuclear compartment identified as the Trans-Golgi Network (TGN) by virtue of its staining for syntaxin 6. This juxtanuclear compartment also stained pos. for rab 7 and for the TGN/pericentriolar recycling endosome marker rab 11, suggesting that the ligand could have been recruited to the TGN from

either late or recycling endosomes. By that time, internalized receptors were detected in Lamp-1-immunoreactive lysosomes. These results demonstrate that neurotensin/NT1 receptor complexes follow a recycling cycle that is unique among the G protein-coupled receptors studied to date, and provide the first evidence for the targeting of a nonendogenous protein from endosomes to the TGN.

REFERENCE COUNT: 73 THERE ARE 73 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

- TI Ligand-induced internalization of neurotensin in transfected COS-7 cells: differential intracellular trafficking of ligand and receptor
- AB The neuropeptide neurotensin (NT) is known to be internalized in a receptor-mediated fashion into its target cells. To gain insight into the mechanisms underlying this process, we monitored in parallel the migration of the NT1 neurotensin receptor subtype and a fluorescent analog of NT (fluo-NT) in COS-7 cells transfected with a tagged NT1 construct. Fluo-NT internalization was prevented by hypertonic sucrose, potassium depletion and cytosol acidification, demonstrating that it proceeded via clathrin-coated pits. Within 0-30 min, fluo-NT accumulated together with its receptor in Acridine Orange-pos., acidic organelles. These organelles concentrated transferrin and immunostained pos. for rab 5A, therefore they were early endosomes. After 30-45 min, the ligand and its receptor no longer colocalized. Fluo-NT was first found in rab 7-pos. late endosomes and later in a nonacidic juxtanuclear compartment identified as the Trans-Golgi Network (TGN) by virtue of its staining for syntaxin 6. This juxtanuclear compartment also stained pos. for rab 7 and for the TGN/pericentriolar recycling endosome marker rab 11, suggesting that the ligand could have been recruited to the TGN from either late or recycling endosomes. By that time, internalized receptors were detected in Lamp-1-immunoreactive lysosomes. These results demonstrate that neurotensin/NT1 receptor complexes follow a recycling cycle that is unique among the G protein-coupled receptors studied to date, and provide the first evidence for the targeting of a nonendogenous protein from endosomes to the TGN.
- ST neurotensin complex NT1 receptor endocytosis intracellular trafficking
- IT Organelle
(coated pit; neurotensin internalization via NT1 receptors proceeds via clathrin-coated pits)
- IT Endosome
(internalized neurotensin/NT1 receptor complexes are initially targeted to endosomes upon import)
- IT Biological transport
(intracellular; neurotensin internalized via NT1 receptors is recruited to trans-golgi network whereas receptors are targeted to lysosomes for degradation)
- IT Lysosome
(neurotensin internalized via NT1 receptors is recruited to trans-golgi network whereas receptors are targeted to lysosomes for degradation)
- IT Neurotensin receptors
RL: BOC (Biological occurrence); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence); PROC (Process)
(neurotensin internalized via NT1 receptors is recruited to trans-golgi network whereas receptors are targeted to lysosomes for degradation)
- IT Endocytosis
(receptor-mediated; neurotensin internalization via NT1 receptors proceeds via clathrin-coated pits)
- IT Organelle
(trans-Golgi network; neurotensin internalized via NT1 receptors is recruited to trans-golgi network whereas receptors are targeted to lysosomes for degradation)
- IT 39379-15-2, Neurotensin
RL: BOC (Biological occurrence); BPR (Biological process); BSU (Biological

study, unclassified); BIOL (Biological study); OCCU (Occurrence); PROC
(Process)
(neurotensin internalized via NT1 receptors is recruited to
trans-golgi network whereas receptors are targeted to lysosomes for
degradation)

=>

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| | ENTRY | SESSION |
| CA SUBSCRIBER PRICE | -1.50 | -1.50 |

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